## RELATIONS AMONG FUNCTIONAL SYSTEMS IN BEHAVIOR ANALYSIS

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This paper proposes that an organism's integrated repertoire of operant behavior has the status of a biological system, similar to other biological systems, like the nervous, cardiovascular, or immune systems. Evidence from a number of sources indicates that the distinctions between biological and behavioral events is often misleading, engendering counterproductive explanatory controversy. A good deal of what is viewed as biological (often thought to be inaccessible or hypothetical) can become publicly measurable variables using currently available and developing technologies. Moreover, such endogenous variables can serve as establishing operations, discriminative stimuli, conjoint mediating events, and maintaining consequences within a functional analysis of behavior and need not lead to reductionistic explanation. I suggest that explanatory misunderstandings often arise from conflating different levels of analysis and that behavior analysis can extend its reach by identifying variables operating within a functional analysis that also serve functions in other biological systems.

Key words: functional systems, scientific explanation, neuroscience, reductionism

Since its inception, the field of behavior analysis has been concerned primarily with variables external to the organism that influence its behavior. Endogenous factors have largely been considered private, inaccessible, and in some cases, hypothetical (Skinner, 1938), a view that persists today (e.g., Barnes-Holmes, 2005; Faux, 2002). These distinctions are contrary to the epistemology of a functional analysis of behavior, which attempts to identify the functions of variables in relation to observable behavior, not their physical locus or ease of accessibility to public scrutiny. Dividing an organism's world into behavioral and biological factors has created counterproductive explanatory problems, often presented as a conflict between reductionism and explanation based on publicly accessible external variables (Moore, 2002). The main purpose of this paper is to suggest that an organism's integrated repertoire of operant behavior has the status of a biological system, similar to other systems, like the nervous, cardiovascular, or immune systems. This collective system of functional behavioral units (see

Thompson & Zeiler, 1986) provides the major mechanism by which organisms interact with, and act upon their physical and social environments. A second purpose is to present evidence that the distinctions between biological and behavioral events are often misleading, since a good deal of what is often viewed as biological (often believed to be inaccessible or hypothetical) can be made publicly measurable using currently available and developing technologies. Finally, I suggest that misunderstandings arise from conflating different levels of analysis and their associated causal relationships.

The integrated repertoire of behavioral units (operants) that have been acquired and maintained under the functional control of motivational or establishing operations, discriminative stimuli, mediating events conjoint with reinforced responses, and consequences (mostly exogenous, some endogenous), functions as a biological system. Embedded within operants are respondents that contribute discriminative stimuli altering the internal milieu, thereby moderating the expression of operant behavior. Individual operants are constituent members of a behavioral repertoire making up that system. In evolutionary terms, the adaptability of these functional behavioral systems and the propensity to construct more complex and higher-order dispositional repertoires accounts for our viability and species uniqueness. These functional behavioral systems perform two impor-

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tant tasks. First, they make it possible to regulate essential physiological and biochemical housekeeping tasks, such as food acquisition, intake and energy balance, liquid consumption which regulates cooling, blood pressure and excretion of wastes via the kidney, and reproduction—abilities all shared with other organisms. Second, our humanness is a product of our ability to acquire and use language, to represent things, concepts, thoughts, or internal stimuli (e.g., emotions) with abstract symbols. As a result, we develop enduring idiosyncratic functional behavioral clusters that contribute to individual uniqueness (see Lubinski & Thompson, 1986), and allow us to function collectively with other people.

The field of behavior analysis has opportunities to expand its reach greatly by exploring variables that interdigitate or overlap with those of other biological systems. In so doing, one must be clear about the relative level of analysis that is the basis for causal attributions. Inclusion of endogenous components within a functional analysis of more complex human behavioral repertoires may provide insights into the ontogeny of larger combinations of interrelated operants that make us unique individuals, as well as enabling us to function collectively as human beings.

#### Biological Systems

Skinner (1972) wrote, "A comprehensive set of causal relations stated with the greatest possible precision is the best contribution which we, as students of behavior, can make in the co-operative venture of giving a full account of the organism as a biological system" (pp. 269–270). A biological system is a functionally related group of interacting, interrelated, or interdependent elements forming a complex whole that serves an organismic function, such as reproduction or endocrine activity. Most biological systems consist largely of endogenous components, such as the heart and blood vessels, or the brain with its neurons and nerve fibers, but such systems also are responsive to external inputs, such as energy (food), oxygen, temperature changes, visual, auditory, tactile, and other external stimulation. Each system operates according to a set of internally consistent and empirically derived principles that have been established, largely independently of one

another. However, most biological systems interact with one another; for example, the vascular system distributes hormones produced by the endocrine system, and endocrine hormones, in turn, can influence vascular function. The nervous system makes it possible for the respiratory system to introduce oxygen into the body, and oxygenated blood permits brain cells to function. The coordinated interactions among various biological systems make it possible for an organism to function as an integrated whole and permit survival.

# A Functional Behavioral System

Within the field of behavior analysis, functional behavioral units have historically been treated as if they were a closed system. The terms, concepts, and defining operations refer back to variables within the system, rather than to components of other biological systems. Just as other biological systems are necessarily interactive, one might more appropriately think of functional behavioral units as a type of dynamical system (Bertalanffy, 1968; White, Harrison, & Mottershead, 1992) interacting with other such units. When a variable in one system also functions as a variable in a second system, it becomes useful to explore the relationships among systems, as suggested by Mach (1914/1959) and Mayr (1982). One system need not be reduced to the other, but exploration of relationships among systems will likely enhance our understanding of each of the individual systems.

In science we are concerned with the degree to which phenomena or events within, say, two systems are concordant, not whether they are identical, since absolute isomorphism across levels of analysis is rare in biology. My purpose in this paper is not to argue that endogenous events (e.g., neurochemical receptor binding) are equivalent in every respect with exogenous operations (e.g., food deprivation), but to examine the degree to which their behavioral properties are concordant.

The functional units of behavior proposed in Skinner's *Behavior of Organisms* (1938) were defined by a complex set of relationships that included four components: (1) motivational events, (2) discriminative stimulus events, (3) responses, and (4) consequences. An operant is an abstraction referring to the relation among these variables that is demonstrated

when the probability of the response component of those relationships is changed by manipulating aspects of the other components (i.e., establishing operations, discriminative stimuli, or consequences). Some of the variables that are components of functional behavioral units can be located beneath the skin, as Skinner (1945) noted many years ago. In the present paper I suggest that endogenous components of operant behavior can be effectively incorporated into a functional analysis, including interactions with other biological systems, to provide a more complete account.

# Interactions among Systems

In his early writing, Skinner (1938) emphasized procedures for identifying regularities among the directly observable environmental variables and changes in the probability of behavior of which they were a function. Although he made reference to physiology throughout his writing (Morris, Lazo, & Smith, 2004), only in his later works did Skinner embrace the idea that objectively measurable events obtained at a different level of analysis could have the status of familiar external variables within a functional analysis. Skinner devoted most of his chapter, "The Nervous System and Behavior", to reasons for rejecting reductionism; however it is noteworthy that he concluded the chapter with a quotation from Ernst Mach's (1914/1959) Analysis of Sensations:

It often happens that the development of two different fields of science goes on side by side for long periods, without either of them exercising influence on the other. On occasion, again, they may come into closer contact, when it is noticed that unexpected light is thrown on the doctrines of one by the doctrines of another (as cited in Skinner, 1938, p. 432).

Mach suggested that overly enthusiastic efforts to combine the two sister fields often fail, and the disciplines eventually go their separate ways. Salient in the present context was Mach's final comment quoted by Skinner:

...the temporary relation between them [the fields] brings about a transformation of our conceptions, clarifying them and permitting their application over a wider field than that

for which they were originally formed. (as cited in Skinner, 1938, p. 432)

Near the end of his book Skinner wrote, "I am not overlooking the advance that is made in the unification of knowledge when terms at one level of analysis are defined (explained) at a lower level" (Skinner, 1938, p. 428). Skinner took pains, as have later theorists (e.g., Moore, 2001; Schnaitter, 1984), to argue that endogenous events are not independent initiators of action; rather, they should be understood as playing a role within the functional analysis of behavior. Endogenous events appropriately enter into a functional analysis when they have concordant functional properties as with those of light falling on cells in the retina or delivery of water to the tongue of a waterdeprived rat following lever pressing.

Theoretical reduction is a popular notion in the biological sciences, but in practice actual reduction across levels of analysis is rare. Marr (1977) pointed out that homogenous reduction, a characteristic of all sciences, occurs when there is a common terminology, such as response, stimulus, reinforcement, and more generally, a common set of variables entering into functional relations as economical descriptions of nature. This, necessarily, is the form of reduction that has occurred within the field of behavior analysis. In contrast, in heterogeneous reduction, terms or concepts that appear within a "lower-level" explanatory theory (e.g., ion flux, long- term potentiation, neurochemical receptor) are lacking in the domain to be explained (behavior analysis) (see, e.g., Nagel, 1961). That renders establishing reduction of one to the other extremely difficult, which is true of the fields of behavior analysis and neuroscience. Batterman (2003) argues that in such cases it is more appropriate to speak of intertheoretic relations rather than reduction (cf. Sklar, 1967). In this borderland between theories and levels of analysis the greatest richness is often found. Instead of seeking to derive logically valid reduction sentences, a concept used by philosophers of science (Carnap, 1938), my goal is to empirically develop bridge hypotheses that contain elements from each level of analysis, thereby permitting probabilistic assertions about the relation of one to the other. To demand identity across levels of analysis is unrealistic and some would say logically impossible

(Feyerabend, 1962). For example, if we ask whether the properties of heat, light, and sound are functionally equivalent in every respect as discriminative stimuli, the answer would be "no". However, the manner in which each variable functions in relation to behavior is highly concordant, though variables differ qualitatively (e.g., visual afterimages) and quantitatively (e.g., delay from stimulus onset until detection). My purpose in this paper is not to argue that endogenous events (e.g., hormones binding to receptors) are equivalent in every respect with exogenous operations (e.g., food deprivation), but to explore the degree to which their behavioral properties are concordant and therefore heuristically and explanatorily useful.

Although many biologists assume theoretical reductionism is their ultimate goal, the historian and philosopher of biology, Ernst Mayr (1982), argued that it is not reduction that should be sought in biological explanation, but rather an understanding of the interactions among biological systems. In this paper the components making up functional units of behavior are advanced as one kind of biological system, some aspects of which are in the external environment and others within the organism. I'll treat such examples as: the effects of establishing operations and history, immediate discriminative stimulus events, characteristics of reinforced mediating events and responses, and their consequences. But the question is not, "Where is a variable physically located?" The more relevant question is, "What role does a variable play in a functional analysis of behavior?" In succeeding sections of this paper, the role of endogenous variables within a functional analysis of behavior will be illustrated.

# ENDOGENOUS COMPONENTS OF OPERANT BEHAVIOR

Motivational Operations

Many organismic states are induced by circumstances that have impinged upon a person that increase or decrease the efficacy of reinforcing events and change the probability of behavior maintained by those events, called setting events or establishing operations. In The Behavior of Organisms (1938), Skinner referred to those events as motivational or emotional operations. Bijou and Baer (1978) wrote, "A

setting event influences an interactional sequence (of behavior and consequences) by altering the strengths and characteristics of the particular stimulus and response functions involved in an interaction" (p. 26). As they used the term, an experimenter or practitioner did not necessarily control a setting event. Michael (1982, 1993) provided a formal definition of Establishing Operations (EOs). In 2000 he elaborated on the EO concept, stating,

The two effects of an EO are an alteration in the reinforcing effectiveness of some stimulus, object, or event (the reinforcer-establishing effect) and an alteration in the current frequency of all behavior that has been reinforced by that stimulus, object or event (the evocative effect). (Michael, 2000, p. 403)

Some transient states that change response probabilities and reinforcer efficacy cannot be linked to identifiable external environmental setting events or establishing operations. Hormonal changes associated with menstrual cycle can change the value of positive and negative reinforcers (e.g., Carr & Smith, 1995) but have no identifiable environmental antecedent. Food is a highly effective reinforcer for people with Prader Willi Syndrome (PWS) who are typically obese, of short stature, and with mild intellectual disability (Holm et al., 1993; Prader, Labhart, & Willi, 1956). Deletion or disruption of a gene or several genes on the proximal long arm of the paternal chromosome 15 or inactivation of that region in individuals who have two maternal copies of the chromosome lead to the cluster of physical and behavioral features making up PWS (Prader-Willi syndrome, 2006). People with PWS have a nearly insatiable appetite, with some affected individuals eating until their stomachs become severely distended with possible perforation (Schrander-Stumpel et al., 2004; Wharton, Wang, Graeme-Cook, Briggs, & Cole, 1997). They have abnormally high levels of a hormone, ghrelin, in the blood stream that produces overeating (DelParigi et al., 2002). The abnormal levels result from a 2to 3-fold increase in ghrelin-producing cells in the stomach and small intestine (Choe et al., 2005). Elevated ghrelin binding to its receptors appears to constitute a sufficient establishing operation to account for the excessive reinforcing effectiveness of food. Self-reports by people with PWS of chronically feeling hungry (e.g., tacting the intereoceptive state), engaging in food-seeking and food consumption are the behavioral consequences of that antecedent peptide-receptor condition.

Neurochemical establishing operations: Sleep deprivation is associated with serotonin and corticosteroid changes in the brain (Anderson, Martins, D'Almeida, Bignotto, & Tufit, 2005; Farooqui, Brock, & Zhou, 1996; Meerlo, Koehl, van der Borght, & Turek, 2002). In a series of studies, Craig Kennedy and coworkers have examined the relationship between sleep deprivation, brain serotonin and its receptors, and avoidance behavior. Compared with baseline, rats' shock-avoidance response rates increased across a range of response-shock intervals when sleep deprived, compared with non-sleep-deprived conditions (Kennedy, Meyer, Werts, & Cushing, 2000). Kennedy and coworkers subsequently examined lever pressing maintained under a multiple fixed-ratio 30 fixed-interval 60-s (mult FR 30 FI 60 s) schedule of appetitive reinforcement in rats exposed to 24, 48, or 96 hr of sleep deprivation and control conditions. Appetitively maintained lever pressing decreased at 96 hr of sleep deprivation but did not change with lesser degrees of sleep deprivation (Kennedy, 2002). Sleep deprivation and serotonin depletion via p-chlorophenylalanine (PCPA) administration resulted in an increase in avoidance responding that was reversed by administration of tryptophan, a serotonin precursor. In a subsequent study, rats were trained to lever press under a freeoperant avoidance schedule. Rats were exposed to 48 hr of sleep deprivation or equivalent control conditions, and then administered a range of dosages of a drug that inhibited serotonin neurons. These conditions also resulted in increased avoidance responding. These effects were reversed by preinjection of the drug that enhanced serotonin neuronal activity (Harvey et al., 2004).

Similar effects of sleep deprivation appear to occur in people as well as laboratory animals. Kennedy and Meyer (1996) studied individuals with developmental disabilities whose daytime problem behavior was negatively reinforced by escape or avoidance from instruction, especially involving task demands. They examined the students' behavior over several weeks using analogue functional analysis methods and

found that increased rates of negatively reinforced problem behavior followed nights during which the students experienced the least amount of sleep. This result does not prove sleep deprivation produced serotonin depletion in these youth, which in turn lowered the threshold for aversively maintained behavior, but the finding is consistent with that possibility. If this analysis is correct, 5-HT1A receptor-binding, often induced by sleep deprivation, may constitute an establishing operation that increases the reinforcing properties of aversive stimuli that are terminated by operant responding. Serotonin has a wide array of other effects as well; however, depletion of this neurochemical appears to mediate sensitivity to negatively reinforced behavior.

# Endogenous Discriminative Stimuli

Neuroreceptor mediated stimuli. Experimental evidence for powerful stimulus control by interoceptive stimuli began emerging in the 1960s, when nonhuman laboratory studies demonstrated convincingly that such stimuli could serve discriminative stimulus functions, much as lights or tones in a traditional operant paradigm. In a prescient study, Schuster and Brady (1971) persuasively demonstrated interoceptive stimulus control over operant behavior. They brought a lever-pressing operant of rhesus monkeys under the stimulus control of the intravenous infusion of epinephrine. Responses produced food reinforcement under a fixed-ratio schedule following epinephrine infusion; but following a saline infusion, lever pressing produced no programmed consequences. The interoceptive stimulus events presumably related to the effects of epinephrine binding at alpha and beta adrenergic receptors (e.g., increased heart rate and blood pressure) set the occasion for lever pressing reinforced by food.

In later studies other researchers demonstrated that a wide variety of drugs producing interoceptive states could reliably serve discriminative stimulus functions (Thompson & Pickens, 1971). Many drugs used to treat mental health problems (as well as those that are addictive) bind to the same chemical receptors in the brain as do naturally occurring neurotransmitters and can also demonstrate discriminative stimulus properties (see Society for stimulus properties of drugs, 2006).

Laboratory studies indicate that animals can reliably respond discriminatively to effects of drugs that mimic normal brain chemical transmitter function. Not only do animals respond discriminatively to consequences of a drug's binding to one brain chemical receptor type versus another (e.g., dopamine versus GABA), they also can distinguish between effects of dosages of the same drug and corresponding internal stimulus states.

The ability to experimentally manipulate interoceptive stimuli pharmacologically that can mimic those produced by naturally occurring neurochemical events lends credence to the notion that endogenous brain chemical changes can also serve as discriminative stimuli. Emotional states, such as anxiety and depression, have discriminative properties that for most people lead us to emit such tacts as "anxious", or "angry" or "sad" as the name for the respective internal states. Those internal events not only have discriminative properties, but they also can modulate the events that could serve as reinforcers. A person with depression may respond to an invitation from a friend to attend a movie by saying, "No thanks, I don't feel like going to a movie tonight". The person with depression is performing a complex conditional discrimination. The friend's verbal invitation is the proximal discriminative stimulus event, but responding to that stimulus is conditional upon the unique interoceptive discriminative stimuli associated with the depressive neurochemical state. When she is experiencing this particular internal state, and a friend invites her to attend a movie, the verbal response that comes to highest strength is to decline the invitation. The depressed individual's utterance is also based on the discriminative properties of the diminished state of strength of her own behavior.

#### Conjoint Mediating Events

As Skinner noted in *Science and Human Behavior*, "The line between public and private is not fixed. The boundary shifts with every discovery of a technique making private events public" (Skinner, 1953, p. 282). Some of those events are conjoint with observable external behavioral changes. The durability of changes in external response probabilities may depend on mediating events that are strengthened conjointly with reinforced responding.

Operant responding that leads to a reinforcing event can be called an effector event or activity. Neurochemical and microstructural brain changes associated with reinforced responding cannot properly be called effector activities because they are not muscular or glandular activities. Instead they are called conjoint mediating events that occur in conjunction with reinforced effector events. Conjoint mediating events occur within the brain subsequent to establishing operations and discriminative stimuli and before reinforced responding. Changes in brain microstructure (e.g., synapse formation) following reinforced responding can be increased via reinforcement and diminished by extinction as illustrated in the following example. Rats trained to perform a reaching task followed by positive reinforcement not only displayed increased percent correct responding of the motor performance but also developed significantly more synapses per neuron than controls within layer V of the caudal forelimb area. In the absence of reinforcement following execution of the reaching response, the number of synapses per cell declined (Kleim et al., 2002). The rats' reaching responses are effector events and the synaptic changes constitute conjointly strengthened mediating events. Having increased the number of synapses in structures involved in emitting a given response increases the probability of recurrence of that response, which thereby lays the foundation for later learning and fluency. Reinforcement not only increased the probability of recurrence of members of a specific operant response class, but also had retroactive effects on the neurochemical and/or microstructural neural events that preceded occurrence of those responses.

Constraint induced therapy. Shaping new responses assumes the organism has an intact nervous system sufficient to enable responding. In 1980 Edward Taub conducted ground-breaking research that he believed would lead to a new treatment for people who were paralyzed by a stroke or other nervous system damage. The experimental work involved severing a nerve to one arm of a rhesus monkey, and then restraining the animal's other (undamaged) arm during recovery. He hypothesized that by differentially reinforcing use of the dysfunctional limb by successive approximations, eventually some or most of

the use of the dysfunctional limb would return. That could only be accomplished by limiting the animal's use of its functional limb. Once sensory input ceased, it was commonly thought that the parts of the cortex that normally received that nerve's input would regress. Around the same time, Kaas, Merzenich, and Killackey (1983) conducted research and reviewed evidence that the brain of adult monkeys undergoes reorganization following loss of sensory input from one limb, such that other brain areas take over the functions of those structures that were no longer connected to the dysfunctional limb. Their work was consistent with Taub's hypothesis. Taub subsequently demonstrated that through repeated reinforcement of use of the dysfunctional limb, partial recovery was possible.

This work led Taub et al. (1993) to develop Constraint Induced Movement Therapy, in which patients with stroke, cerebral palsy, and other neuromotor disorders substantially recovered if they arduously practiced using their dysfunctional limb following injury. He demonstrated that some of the long term, even permanent, motor impairment following nervous system damage is due to a combination of motivational and learning factors resulting in a "learned non-use" rather than being the result of the CNS damage per se. A functional analysis of his rehabilitative work with monkeys and later with human patients was difficult to rationalize unless one assumed it was possible that repeated use of the dysfunctional limb would produce significant changes in the brain-and these are now known to occur (see, e.g., Pons et al., 1991).

Intensive Early Behavior Therapy (IEBT) for children with autism is the only known psychological or behavioral treatment that produces sustained changes in a severe behavior disorder following treatment. Lovaas's (1987) demonstration that approximately half of the children with autism diagnoses develop social and language skills that are sustained following treatment, and permits them to function intellectually in the average range, is compelling testimony to the power of behavior analytic principles and procedures. However, half of the children in Lovaas's and later studies fail to show such improvement. Recently Sallows and Graupner (2005) found that children with lower baseline IQ's, lack of joint attention, and little or no motor or verbal imitation (at 2 to 3 years of age) and with autism diagnoses showed limited improvement in core autism symptoms over 4 years of 30+ hr per week of IEBT. That raises the question of why IEBT is effective with some children with autism but not with others. Whatever is different about the brain functioning of children with autism must be consistent with the IEBT evidence, namely that half of the children dramatically improve and half show limited gains using what appear to be the same behavior therapy techniques.

Bauman and Kemper (1994) found that brain tissue samples from young children with autism had an abnormally large number of tightly packed immature neurons in limbic and prefrontal areas as well as cerebellar vermis, with few synapses. By adolescence or young adulthood many neurons had disappeared, but those that remained had rich dendritic arborization and were synaptically connected. This suggests that some of the neurons in those structures had the potential to form synapses, but presumably because they had inadequate input during development, neuronal pruning led to their regression.

I recently suggested that children with autism diagnoses who were responsive to IEBT had the potential to form synapses in brain areas known to be dysfunctional in autism, whereas those who profited far less from IEBT had abnormalities in the same structures, but were not amenable to correction by forming new synapses (Thompson, 2005). IEBT involves repetitive training that can be accomplished only by enlisting the active engagement of those specific brain structures. For example, a child cannot anticipate the events that are likely to occur during a sequential social interaction without the active engagement of cells in the orbitofrontal cortex. By repeatedly requiring the child to perform such discriminative tasks, new synapses will very likely be formed in that brain area. The more those skills are practiced, the more functionally consolidated or effective those synapses become, which is very likely why the effects are permanent. In the absence of practice, as was common in the past when young children with autism were placed in sequestered residential or ineffective school settings, synapses failed to form, and the neurons that would normally have served that function regressed through disuse. Those structures became permanently nonfunctional.

Much research shows that synapse formation can be activity-dependent (Cohen-Cory, 2002; Greenough & Churchill, 2003; Jontes & Smith, 2000). Errors in brain development in some human disabilities are associated with activitydependent retraction of dendritic spines, which may be insufficient (Boulanger & Shatz, 2004). Dendritic spines are the locus on certain brain neurons where synapses are formed. Nonfunctional dendritic spines are excessively long, and fail to convey synaptic currents from the head of the spine to the dendrite (and cell body) to which the extralong spine neck is attached. In normal brain tissue, dendritic spines change continuously through typical use and disuse. The spines contract with mechanisms similar to muscle: actin and myosin. Early disuse could reduce expression of the molecules needed for spine contraction, which is required to make the spine functional. By identifying the genes that produce the proper amount of substrate required for dendritic spine contractions that are only turned on by performing specific tasks, one has a more complete account of how synapse formation does or does not occur, and of the role of functional behavioral units in that process.

To the degree that functional behavior analysis procedures promote discriminative responding that can only be accomplished by causing release of neurotransmitters in cells in structures that are synaptically deficient, one system, the four-component operant (establishing operations, discriminative stimuli, conjoint mediating events, and maintaining consequences), interacts with another functional system (synaptogenesis). The promotive effect of training does not imply that the fourcomponent operant is reduced to synaptogenesis. Rather, it suggests that a conjoint mediating event (synapse formation and consolidation) becomes a component of the fourelement operant. The point of contact between the two systems is the activity-dependent release of neurotransmitters that promotes synapse formation.

One need not refer to correlated brain changes (synaptic-reinforced mediating events) in order to conduct IEBT with young children with autism. However, failing to do so leaves the former question unanswered: "Why do some children greatly benefit from IEBT and others do not?" Those who do not benefit equally

from IEBT may have damage to some of the same brain structures, but the damage may have been produced by a different mechanism, one that is not amenable to amelioration by forming and consolidating new synapses. Axons could have been misrouted to the wrong structures, or receptors necessary for forming synapses could be damaged, as happens due to toxin exposure or some genetic defects (e.g., in Fetal Alcohol Syndrome, GABA receptor gene deletion in Prader Willi Syndrome). This possibility does not imply that children who are minimally responsive to IEBT should receive no intervention. However, it suggests that we must understand better the mechanisms underlying their disability and determine which intervention strategies are most likely to ameliorate those limitations.

# Maintaining Events

Drug dependence. Throughout the first half of the 20<sup>th</sup> century, the medical community assumed that drug addiction was a purely physiological or biochemical process. Pharmacologists and other medical researchers commonly believed that addiction could be characterized in terms of the transaction between a drug molecule and the tissue upon which it impinged. In the early 1960s that conceptualization changed when experiments in laboratory animals indicated that opiate drugs could serve as reinforcers for operant behavior—very much like more familiar reinforcers such as food or water (Thompson & Schuster, 1964; Weeks, 1962). Reinforcement schedules, stimulus control procedures, and other typical contingency manipulations affected drugmaintained behavior in ways that were largely indistinguishable from behavior maintained by other consequences (Pickens & Thompson, 1968; Schuster & Thompson, 1969). These and other subsequent studies suggested that understanding addiction required explication of the relationships among a drug's biochemical and physiological effects and their interaction with reinforcement contingencies. Performances in these animal models are highly predictive of which newly developed drugs are mostly likely to have addictive properties in people (Ator & Griffiths, 2003; Higgins, Heil, & Lussier, 2004; Meisch 2001; Vanderschuren & Everitt, 2005). As a consequence, operant drug self-administration procedures are now required by the U.S. Food and Drug Administration and are used world wide to screen for the abuse-liability of newly developed drugs.

Over the intervening years numerous studies have demonstrated that when most drugs are self-administered by laboratory animals, those agents bind to specific brain receptors that mediate the reinforcing effect of the drug. If those receptors are blocked by an antagonist, the drug no longer serves as a reinforcer, and previously drug-maintained operant behavior extinguishes (Yokel, 1987). But addiction does not reside solely in drug-receptor binding. Whether drug administration serves as a maintaining event also depends on the nature of the contingency relationship between responses and their consequences. Spealman (1979) trained squirrel monkeys to self-administer cocaine by pressing a lever under a variable-interval (VI) 3-min schedule of reinforcement. Concurrently, the monkeys could terminate the stimulus indicating the availability of self-administered cocaine by pressing a second lever where under a FI 3min schedule, a 1-min timeout was presented. The monkeys reliably terminated the opportunity to self-administer cocaine under the fixed-interval schedule and continued to selfadminister cocaine under a variable-interval schedule at other times. The maintenance of behavior by schedule-controlled drug injections and by termination of access to drug injections indicated that whether and in what way a drug would control behavior depended on the contingency relationships, even with the same highly addictive drug.

Self-injury in developmental disabilities. Selfinjurious behavior by people with developmental disabilities shares features in common with behavior maintained by drug self-administration. People who engage in self-injury do so for various reasons. Functional analysis and assessment research has demonstrated that self-injury most often serves a demand-avoidance function or leads to access to caregiver attention (Khang, Iwata, & Lewin, 2002). For some people with severe and unremitting selfinjury, there is evidence that such behavior is maintained at least in part by the neurochemical consequences of self-inflicted pain (Sandman, Hetrick, Taylor, & Chicz-DeMet, 1997), much as drug-maintained behavior is reinforced through the mediating brain chemical actions of the drug that has been self-administered. Among the consequences of selfinflicted painful stimulation (e.g., following self-injury) is release of beta endorphin, an analgesic peptide that binds to the brain's opiate receptors, the same receptors to which morphine and heroin bind. Cataldo and Harris (1982) hypothesized that some selfinjury may be maintained by endogenous opioids released following self injury. Thompson, Hackenberg, Cerutti, Baker, and Axtell (1994) and Sandman et al. demonstrated that when people with chronic self-injury were treated with the opiate antagonist drug naltrexone, between 30 and 50% of individuals stopped or markedly reduced self-injury. Sandman and colleagues obtained blood samples within 5 min following an episode of selfinjury and determined the blood levels of beta endorphin relative to baselines when no selfinjury occurred. They then treated the same individuals with naltrexone and found a 0.67 correlation between the amount of beta endorphin increase following self-injury and the amount of reduction in self-injury produced by naltrexone. It appears that for such individuals, a portion of the maintaining consequence for self-injury is binding of beta endorphin to the brain's opiate receptors, which can be blocked by naltrexone, much as cocaine self administration can be reduced by administering specific dopamine receptor antagonists.

Self-injury is usually multiply determined. In treating a 14-year-old boy with significant selfinjury, Symons, Fox, and Thompson (1998) conducted an analysis of the effects of naltrexone alone and combined with behavioral interventions. The latter included augmentative communication training for requesting escape from nonpreferred tasks, augmentative communication training for requesting teacher attention, and augmentative communication training alone. Using an ABCBC design, they demonstrated that while naltrexone alone (Condition B) reduced self-injury by half, adding augmentative communication intervention (Condition C) reduced self-injury by 95–100% in the classroom during the C intervention phase. This result suggests that the youth's self-injury was maintained by a combination of positive and negative social reinforcement and release and binding of beta endorphin to his brain's opiate receptors.

## DISCUSSION

# Causal Analysis

Mach argued that scientific explanation is "condensed indirect description", by which he meant causal explanation involved not only describing what was observed but providing an account of the observed events in terms of more general principles derived from other observations (see discussion by Marr, 2003). The term cause as used in this article refers to an event, process, or microstructural change shown to be at least probabilistically related to a given effect. This meaning of cause is in the tradition of Reichenbach's (1956) and Hitchcock's (1993) probabilistic causality theories and implies a temporal directional arrow of causality. Dretske (2004) proposed distinguishing triggering from structuring causes. According to this formulation, structuring causes are events that, in turn, cause a trigger to produce its effect. Structuring causes are higher-order dispositions (see below) that make triggering causes possible. Establishing operations and reinforcing events are structuring causes that make reinforcement possible and increase the probability of recurrence of operants. Discriminative stimuli are triggering causes in this formulation.

Food deprivation is an establishing operation that increases the reinforcing value of food. General causal claims, such as "food deprivation causes food to serve as a reinforcer," refer to repeatable general properties of causes and effects. Singular causal statements refer to specific events that have spatiotemporal locations, such as "food deprivation caused food to serve as a reinforcer for lever pressing by Rat 287 in operant chamber four." There is also a difference between the cause and a cause, a distinction that originated with Mill (1843/ 1986). Food deprivation has widespread physiological and biochemical effects, including changes in blood sugar, glucagons, amylin, ghrelin, cholysystokinin, and neuropeptide Y (e.g., Beck, 2006; Moran, 2006). It is accurate to say that food deprivation is a cause of the reinforcing value of food, but if it could be experimentally shown that effects of food deprivation on the reinforcing value of food were uniquely mediated by one or some combination of these biochemical changes, then it would be more parsimonious to say that the magnitude of the reinforcing properties of

food are regulated (i.e., caused) by *the* specific biochemical events that had been identified. In the present article I have employed a probabilistic analysis of causes, without making claims that they are *the* cause of specific effects. I have been primarily concerned about the degree to which endogenous events demonstrate probabilistic causes concordant with those of established environmental variables within a functional analysis of behavior.

# Dispositional Analysis

Ryle (1949) contended that mental states could be analyzed as dispositions, that is, a propensity or predisposition to behave in a particular way under specified circumstances (Cross, 2005). He warned against what he called "category mistakes," that is, ascribing a property to a state that it could not possibly have. Ryle meant that a mental (i.e., a non-physical event) could not cause a behavioral event (a physical event), and language and concepts applicable to one are not appropriate for the other. We say "I thought very hard about it" or "I'm drawing a blank", metaphorically applying terms from the physical world to mental activities. Ryle argued that applying physicalistic language to phenomenological events leads to illusory consequences, a problem that plagues much of psychology (Holth, 2001).

Although Ryle (1949) suggested that one need not look for causal or mechanistic explanations of the dispositions, later writers have largely disagreed (Mellor, 1974; Mumford, 1998). The causal basis of an object's disposition is something like a microstructural property of the object that is causally responsible, under specifiable conditions, for the manifestation of the disposition (Prior, Pargetter, & Jackson, 1982, p. 251). According to this reasoning, a statement such as, "A glass vase breaks when struck by a hard object because of its irregular silicon dioxide molecular structure," would provide a more adequate dispositional account than "A glass breaks because it is fragile." The latter account appears tautological, though many would contend it is not because it can be reduced to further dispositional properties or mechanistic explanans (e.g., Mellor, 1974; Mumford, 1998; Prior et al., 1982). A tautological dispositional explanation becomes scientifically meaningful when the explanatory disposition can be linked to other lawful relationships of a more general sort. Vanderbeeken and Weber (2002) argued that dispositional explanations can be useful in behavior analysis. As with the glass vase example, it would be unacceptably circular to assert that a rat's lever pressing increased in frequency because it was reinforced, unless the concept of reinforcement can be tied to a broader set of generalizations and abstract concepts (e.g., schedule effects, matching, contrast effects, reinforcement's role in the abstract construct "operant") (see Meehl, 1950).

A lay person might ask, concerning a person she is observing, "Why is that man reporting that he sees and hears people talking who no one else sees, and appears to be talking to people who are not apparent to others?" A clinical psychologist might reply, "Because he has schizophrenia". The dispositional causal statement that people who have schizophrenia have the disposition to hallucinate is not simply reification, because it refers to one of a complex set of interrelated dispositions that comprise the properties of the condition called schizophrenia.

A more satisfactory dispositional account calls for a description of conditions that give rise to the disposition to hallucinate and engage in psychotic verbalizations. Different accounts may be provided, such as from data suggesting hallucinatory behavior is under immediate interoceptive stimulus control (Leibman & Salzinger, 1998; Salzinger, 1973) and that it is possible to reduce psychotic verbalizations by extinction and differential reinforcement of appropriate speech (Ayllon & Haughton, 1963; Leiberman, Teigen, Patterson, & Baker, 1973) or, alternatively, that hallucinations arise from hyperactive dopaminergic signal transduction (Sedval & Farde, 1995). The former refers to a structuring cause of the disposition whereas the latter refers to triggering neurochemical causes responsible for the current disposition to hallucinate. Within the field of behavior analysis, the former types of dispositional accounts have predominated, though at times there may be advantages of considering endogenous causes as well.

Once we know that a system has a certain disposition, we know how this system will behave in certain situations due to the presence of an underlying set of properties that functions as a set of internal causal factors (i.e., an organism's state resulting from a reinforcement history or a neurochemical condition). Dispositions do not tell us which causal basis is to be found in a system, they tell us that there is some sort of causal basis that, together with certain triggering situations, will bring about typical behavior (Vanderbeeken & Weber, 2002).

# Dispositional Misattribution

As Meehl (1993/2006) pointed out, explanatory problems can arise when we propose an account at a given dispositional level that may not apply to that level, but is appropriate to another dispositional level. A common example is a statement such as "genes cause behavior". Genes cannot cause behavior. Genes produce proteins. Proteins are components of higher-level dispositions that may eventually influence, but not cause behavior. Genes may be necessary prerequisites for particular behavioral events to occur, but their expression depends stochastically on experience and other epigenetic factors.

At times similar mistakes are made in attributing dispositional causes of behavior to external environmental events. Staff members working in group homes or classrooms may ask, "Why do our residents (or students) with intellectual disabilities engage in seemingly unpredictable behavioral outbursts (e.g., aggressive, disruptive, or self-injurious behavior)?" Therapists and teachers collect data indicating that behavior outbursts seldom if ever occur on some days, but do occur with high frequency on others. They then conduct a functional analysis of putative proximal controlling variables and find that on days with frequent outbursts, behavior problems serve as task-avoidance responses terminating demands (Kennedy et al., 2000). On days with few outbursts, similar demands fail to produce outbursts. If we then further ask why the rates of such avoidance behavior fluctuate from day to day in apparently unpredictable ways, we find that the threshold for demand avoidance covaries with the number of hours per sleep the previous night. Evidence from laboratory animal studies suggest sleep deprivation may lead to depletion of brain serotonin (Harvey et al., 2004), which in turn lowers the avoidance threshold. The assumption that a functional analysis of proximal environmental controlling

variables provides an adequate account overlooks the possibility that the origins of the disposition in question (propensity to engage in avoidance behavior) are to be found at a different dispositional level, neurochemistry. The initial point of intervention might involve rectifying the sleep abnormality by administering a serotonergic medication like trazadone. Once that has been accomplished, a functional-analysis-based social intervention will more likely be effective on the remaining occasions on which behavioral outbursts occur.

Figure 1 presents a hypothetical causal chain of events leading to signs and symptoms of autism that illustrates levels of analysis and orders of dispositions. In thinking about causes (i.e., what causes what) one should be clear about the level of analysis or order of disposition being discussed. Obviously, from this hypothetical analysis, there are potentially several levels at which intervention might occur to prevent or ameliorate the development of autism symptoms, from gene therapy to pharmacological interventions targeted at dendritic spine activity to Early Intensive Behavior Therapy (IEBT). Actions taken at a given dispositional level cannot logically change higher-order dispositions (i.e., IEBT cannot cause new genes to be produced), but can affect lower-order dispositions (e.g., selective synapse formation).

Much as the statement "genes cause behavior" involves a dispositional misattribution, behavior analysts commit similar mistakes when they assert that autism is learned through differential reinforcement of autistic behavior (Drash & Tudor, 2004). Autism is not a set of behaviors to be learned. Specific operants such as hand flapping and tantrums could likely be acquired through differential reinforcement. However, autism is a complex state, expressed in an array of specific features present at various levels. These include objectively measurable dysfunction in five or six brain areas, serotonin abnormalities, frequent errors on human Chromosomes 2, 7, 13, or 15, lack of eye contact apparent shortly after birth, lack of social initiations from infancy (e.g., gestural, tactile), inability to speak so as to communicate effectively, excessive and qualitatively unusual stereotyped behavior within the first year of life, intolerance for changes in external stimulus conditions leading to tantrums-some or all of these characteristics

detectable by 12 to 18 months of age. X (differential reinforcement of hand flapping) could not cause Y (lack of amygdala activation when looking at faces). Differential reinforcement of hand flapping cannot logically cause the brain abnormalities or the covarying cluster of behavioral features seen in autism.

Thinking more clearly about elements in such a causal sequence is necessary to prevent our making unfortunate category mistakes, and can enlighten us about the relative effectiveness of different foci as well as techniques of intervention. It is illogical to suggest that administering an atypical antipsychotic drug (Roeder, 1995) or repeatedly stroking the child's skin with a brush as part of Sensory Integration Therapy (Kranowitz, 2005) would replace or overcome the consequences of the missing synapses or substitute for appropriate and necessary learning experiences. However, given a condition of limited synaptogenesis, if we have reason to believe that intensive early behavior therapy could promote synapse formation as indicated in Figure 1, then such would seem a rational strategy.

# Superordinate Dispositions

The field of behavior analysis has provided detailed accounts of lower-level behavioral dispositions, and to some degree, higher-order dispositions, though with limited specificity. For example, we have some understanding of the establishing operations, stimulus control, response characteristics, and consequences that might lead a child to raise her hand in the classroom. But we would be hard pressed to prescribe the necessary and sufficient conditions to create a "needy" or a "resilient" child, terms that could be translated into objective behavioral descriptions. We may have reasonable hunches, but these hunches would merely be plausible accounts, not the types of functional analyses that have been provided first- and second-order dispositions. Higher-level dispositions are likely to be acquired through relational learning processes (e.g., Sidman, 1994). Understanding the conditions giving rise to superordinate categorical dispositional clusters has practical as well as theoretical implications. McIlvane and Dube (2003) have suggested that generalized stimulus control by more complex and formally dissimilar stimuli depends on the agreement

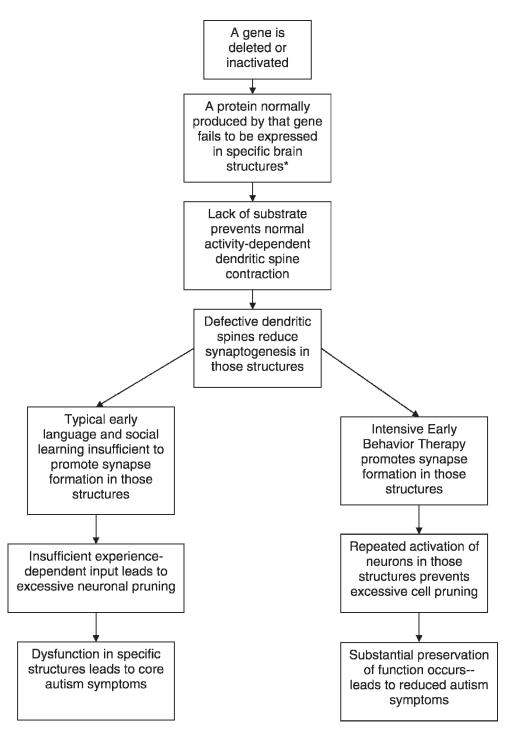


Fig. 1. Hypothetical causal chain leading to signs and symptoms of autism, illustrating orders of dispositions. Autism symptoms and features constitute a first-order disposition. Defective dendritic spines reducing synaptogenesis impair the ability to learn typical language and social skills, a second-order disposition. Lack of substrate for activity-dependent dendritic spine contraction is a third-order disposition. Lack of protein expressed in specific brain structures is a fourth-order disposition. Absence of a gene to produce those proteins is a fifth-order disposition. Early language and social learning experience is an event that impacts synapse formation, a second-order disposition.

between putatively relevant aspects of those stimuli, as perceived by the person impacted by those stimuli. They refer to this relationship as *stimulus-control topography coherence*. McIlvane (2003) has suggested that providing an adequate account of these processes may require new behavioral principles. Hayes and co-workers (Hayes & Barnes-Holmes, 2001) have proposed a related approach (Relational Frame Theory) in an attempt to account for ways in which apparently dissimilar stimuli become linked into elaborate verbal response classes.

Information making it possible for psychotherapists to dissect a client's superordinate relational stimulus-response classes may be instrumental in resolving personal problems. A client who has repeatedly lost jobs because of confrontations with supervisors may be aided by strategies for devolving a stimulus class such as "authority figures", with their presumed properties, and establishing more relevant stimulus-response classes. Attempting to change racial and ethnic stereotypes by differentially reinforcing young children for saying positive things about their peers from different backgrounds may have little impact; while perhaps laudable, such a procedure may not be directed at the appropriate dispositional level. Resolving racial and ethnic discriminatory behavior may depend on better understanding how such superordinate classes (e.g., Black, Caucasian, and Latino) are established, and how they may be changed to incorporate more socially relevant personal attributes.

## CONCLUSION

I have suggested in this paper that the concatenated repertoire of operant behavioral units (with embedded respondents) comprises a functional biological system comparable to other biological systems. These behavioral dispositions are hierarchically organized beginning with individual response classes that are combined to create increasingly complex repertoires that distinguish human symbolic behavior. There is nothing inherent in a functional analysis of behavior that requires all of the variables to be located external to the skin. Indeed, as is true of other biological systems, there may be advantages of studying interactions between and among functional behav-

ioral repertoires and other systems (e.g., nervous, endocrine, and immune systems). The four basic components of operants motivational operations, antecedent stimuli, responses and their associated mediating events, and reinforcing consequences—may include endogenous conditions as well as events occurring outside the skin. In some instances substantial overlap between behavioral processes and endogenous physiological and biochemical events may occur, but a complete reduction of one to the other may likely prove difficult, if not impossible. However, providing more information regarding the series of events occurring within the nervous system between presentation of a discriminative stimulus and emission of a response can play an important role in completing the account (Skinner, 1989).

The proposed analysis addresses several problems. For behavior analysts, the proposed approach makes it largely unnecessary to ask whether the cause of a specific instance or class of behavior is environmental or biological. This distinction is not helpful; rather the approach presented provides a systematic way of formulating experimental questions regarding the role of endogenous variables within a functional analysis. For example, what is the behavioral mechanism by which an antidepressant medication changes the behavior and psychological functioning of a person with a major depressive disorder? Does it reduce the negatively reinforcing value of social interactions as well as enhance the value of putative positive reinforcers, or does it change the interoceptive discriminative stimuli inherent in a depressive state? The proposed strategy assists us in thinking about the proper level at which to intervene to produce a given effect, or how to best measure a treatment effect, or, more generally, allows us to examine a problem of interest with greater care.

For neuroscientists and neurogeneticists, the proposed analysis assists in thinking more clearly about the causal role of genes vis à viz behavior, as well as neuropathological conditions expressed as behavioral or developmental disorders. The approach may, for example, facilitate identification of *endophenotypes*. Gottesman and Shields (1967) and Gottesman and Gould (2003) argued that identifying specific physical features (often physiological or neurochemical) that uniquely covary with

a clinical syndrome is more likely to be productive in unraveling gene-psychopathology relationships than focusing on clinical symptoms (e.g., thought disorder) that are often influenced by experience. The use of behavior-analytic strategies could extend the range of highly reproducible phenotypic features that could be included in endophenotypic research strategies. For example, the degree to which discriminative responding to facial expression could be established in very young children could be a predictor variable in heritability studies of autism spectrum disorders.

The biologist Waddington (1942) coined the term epigenetics, referring to modifiable heritable changes in gene function that occur without a change in DNA sequence (genotype). These changes may be induced spontaneously, in response to environmental or experiential factors, or to the presence of a particular allele. The degree to which modifiable epigenetic changes occur depends on the underlying mechanism. The approach suggested here could provide neuroscientists with more sophisticated approaches to exploring activity-dependent neural processes. Behavior-analytic laboratory procedures may make it possible to explore specific experience-dependent epigenetic influences that may alter gene expression in specific brain areas. Early differential reinforcement of discriminative responding to visual images of hand and arm movements may promote gene expression in the mirror neuron system, for example.

Finally, clear thinking about dispositions causally linked to individual performances and larger behavioral classes may help avoid errors of causal attribution: what Ryle called category mistakes and what Meehl described as errors of dispositional levels. Avoiding such mistakes can also affirmatively guide more productive analyses and intervention strategies.

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